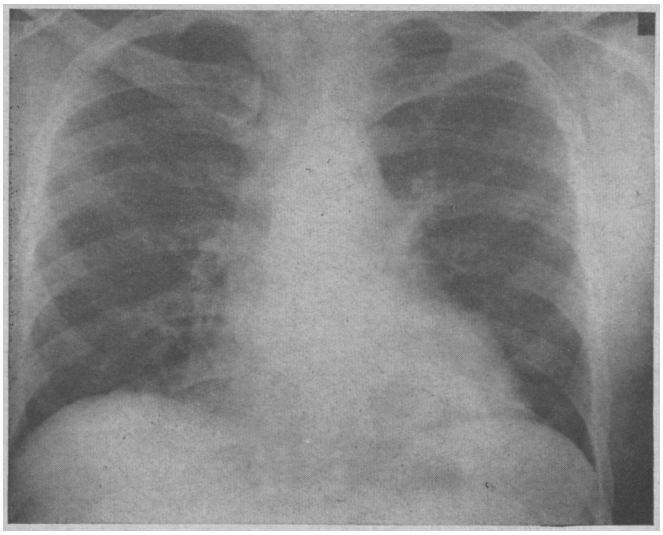
cant fibrosis has developed.[2,3] Even under these circumstances, some patients with this disease have not responded in any way to the use of corticosteroid therapy,[4,5] while others have experienced symptomatic improvement without objective benefit.[6] In some instances, cessation of the administration of steroids has been followed by uncontrollable relapse and, ultimately, the death of the patient. Other patients have responded to treatment, with symptomatic improvement accompanied by clearing visible on roentgenograms and measured improvement in pulmonary function,[7] similar to the response of our patient.

Although these indications of improvement are helpful, direct examination is the only reliable method of determining tissue response to treatment. The present case is of interest because, to the best of our knowledge, it is the first in which biopsy material has been obtained before and during steroid therapy. The core of tissue obtained on needle biopsy was generous and was thought to be representative of a large portion of a lung which had been involved by a diffuse process.

When the dose of steroid was raised to a high level, and its use increased to a daily basis, the patient's condition began to improve. This improvement in his clinical and physiologic status has been maintained for 30 months, and he has not experienced any limitation in his activity, despite discontinuation of his medication 14 months before his last tests of pulmonary function were performed. The optimal dose of corticosteroid is presently unknown, but the sequence of events in this case would suggest that high doses may be helpful when initial levels used for treatment seem ineffective.

While improvement in the patients with interstitial pulmonary disease may occur without steroid therapy, the sequence of events in this case suggests a beneficial effect of the steroid after the dose was increased. Collagen is no longer regarded as inert, and metabolic turnover is known to occur.[8]

4. Almost-complete clearing of pulmonary parenchymal infiltrates in March 1967, at time of percutaneous needle biopsy of lung.



We believe the correlation of tissue, physiologic, and roentgenographic improvements in this case support the concept of potential reversibility of this syndrome.

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Methanol Poisoning

Karl Closs, MD, and Claus O. Solberg, MD

A 63-year-old man consumed a nonidentified beverage 18 hours before hospitalization. His symptoms—headache, nausea, vomiting, abdominal pain, drowsiness, and marked dyspnea leading to deep coma—suggested methanol poisoning. Pupillary reaction was impaired, acidois was present, and the urine contained methanol, formic acid, and formaldehyde. Treatment with sodium bicarbonate, saline, and glucose-insulin was immediately instituted. Oliguria was treated with extracorporeal dialysis, and the patient recovered completely except for a thrombosis in the left leg.

THE REASONS for presenting an isolated case of methanol poisoning are twofold. First, the present case is unusual; despite severe poisoning, with excessive acidosis and oliguria lasting one week, the patient recovered without any permanent sequelae. Second, during the acute phase of the intoxication, a larger amount of formaldehyde than normal was demonstrated in the urine.

Report of a Case

A 63-year-old mill worker was admitted to the hospital as an emergency case, with undiagnosed coma. The patient had not previously been hospitalized, nor had he consulted

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doctors, but a history of many years of excessive use of alcoholic beverages was elicited. According to his wife, he had drunk a bottle (1 liter) of what she thought was beer about 18 hours before admission to the hospital. In the middle of the night, six hours after the ingestion of the drink, the patient woke up. He appeared to be inebriated, and complained of headache, nausea, and abdominal pain. Vomiting was pronounced during the next two hours, but later the patient became drowsy and weak, with deep respiration and increasing coma.

On examination, he was deeply comatose, with no response to pain stimuli, and complete absence of tendon, abdominal, and plantar reflexes. Respiration was very deep and frequent, and the patient was dehydrated. Forward flexion of the head was slightly reduced. Ophthalmoscopic examination revealed mild hyperemia of the disk and retina. The pupils were dilated, with impaired reaction to light. Body temperature was 98.2 F (36.8 C); blood pressure, 140/95 mm Hg; and pulse rate, 120 beats per minute. There were no other abnormal physical signs, and no signs of infection.

Urinalysis on admission gave the following values: specific gravity, 1.008; pH, 5; proteinuria of 0.08%; slight hematuria; and no glycosuria or ketonuria. Blood tests revealed the following values: white blood cell count (WBC), 33,500/cu mm; hemoglobin, 16.8 gm/100 ml; sedimentation rate, 3 mm/hr; urea, 68 mg/100 ml; and glucose, 155 mg/100 ml. Serum values included pH, 6.95; total carbon dioxide (CO_2) combining power (alkali reserve), 5.7 mEq/liter; arterial carbon dioxide pressure (Pco_2), 22.5 mm Hg; sodium, 137 mEq/liter; potassium, 7.7 mEq/liter; and chloride, 100 mEq/liter. Examination of cerebrospinal fluid (CSF) gave the following values: cells, 4/cu mm; protein, 180 mg/100 ml; glucose, 140 mg/100 ml; and chlorides, 145 mEq/liter. The electrocardiogram showed changes compatible with hyperkalemia. Later, methanol, formaldehyde, and an increased quantity of formic acid were found in the urine.

Treatment was started at once, and during the first five hours the patient was given intravenously 1,000 ml of 2.6% sodium bicarbonate solution (500 ml was administered during the first 15 minutes), 1,000 ml normal saline solution, and 1,000 ml of 5% dextrose solution with 20 international units (IU) of insulin. After this, clinical improvement was apparent. The patient could move his arms and legs a little, and tendon reflexes were present, but weak. Respiration was no longer so deep and frequent, but his pupils were still dilated, though they showed sluggish reaction to light. The serum pH had increased to 7.19, and the alkali reserve to 9.5 mEq/liter. The serum potassium level had fallen to 6.0 mEq/liter.

During the next eight hours, the patient received 1,350 ml of 1.3% sodium bicarbonate solution, 1,000 ml of normal saline solution, and 1,000 ml of 5% dextrose solution with 20 IU of soluble insulin. Clinical improvement was now pronounced. The patient could move around in bed and talk a little. However, he was delirious, and gave completely inadequate answers to questioning. His pupils reacted better to light, and vision did not seem to be completely destroyed. Since he was able to eat and drink, intravenous fluid administration was discontinued, and sodium bicarbonate was given by mouth. After this treatment, laboratory findings were as follows: serum pH, 7.35; alkali reserve, 17 mEq/liter; serum Pco_2, 30.5 mm Hg; sodium, 144 mEq/liter; potassium, 4.8 mEq/liter; and chloride, 106 mEq/liter.

The patient was given a total of 6,350 ml fluid intravenously during the first 13 hours in the hospital, but his urine output was only 130 ml. The following day, urine output fell to 50 ml and the blood urea value rose to 157 mg/100 ml. Forty-eight hours after admission to the hospital, hemodialysis was started, and repeated seven days later because of persistent oliguria and a blood urea level of 308 mg/100 ml. During the following two weeks, urine output increased to 3,800 ml/day, and then dropped to a normal level after another week.

Three days after admission to the hospital (the day after the first hemodialysis) pupil reflexes were normal, but vision was slightly reduced. A week later, ophthalmologic findings were normal.

Except for a thrombosis in his left leg, the patient made an uneventful recovery. He stayed in the hospital for 30 days. Physical examination six months later revealed nothing abnormal. By then, he had also stopped drinking alcoholic beverages.

Comment

The present patient showed all the symptoms typical of methanol poisoning, including excessive acidosis. The finding of methanol and formic acid in the urine substantiated the diagnosis. There is no reason to suppose that the patient had ingested any other toxic substance, such as ethylene glycol, which could have explained the acidosis and renal effects, or methyl chloride, which could have explained the isolation of methanol and formic acid in the urine.[1] In the latter kind of intoxication, acidosis is usually moderate, and there is no impairment of vision. However, oliguria is uncommon in methanol poisoning, although it has occasionally been observed. Degeneration of the epithelium of the convoluted tubules is said to be an almost constant finding in methanol poisoning,[2] and changes in renal function have been described in fatal cases.[3] But to our knowledge, severe oliguria such as our patient had, and recovery without permanent sequelae have not been reported.

It has been assumed that the diagnosis of methanol poisoning could be verified by the demonstration of formic acid in the urine. This substance is, however, a normal constituent of urine. Therefore, as in our case, a quantitative determination of urinary formic acid excretion must be performed. This procedure is rather tedious, and, without practice, reliable results are difficult to obtain. It is much easier and less time-consuming to show qualitatively that methanol is present in the urine or blood, or both. Therefore, in order to make an early diagnosis of methanol poisoning, the isolation of methanol should be requested. The determination of formic acid value should only be used as a supplement and performed by experts in cases where 24-hour collections of urine are available, ie, for patients in the later stages of intoxication.

It is now generally accepted that neither methanol itself nor formic acid is responsible for the pathological changes—particularly those in the retina—demonstrable in cases of human methanol poisoning; the so-called proximal toxic agent is formaldehyde. Although the paramount role of this agent in the pathogenesis of the ocular symptoms is quite firmly established experimentally, the metabolic route by which methanol is transformed to formaldehyde is still a matter for dispute,[4] and presence of formaldehyde has not been demonstrated unequivocally in cases of methanol poisoning. In the present case, formaldehyde was sought in the distillate from the acidified urine. The distillate gave a stronger reaction with the chromotropic acid reagent used than distillates

from normal urine did. Therefore, formaldehyde should be sought in the urine of other patients with methanol poisoning.

Treatment should include administration of large doses of bicarbonate and sufficient doses of ethanol until the alarming visual symptoms have subsided and acidosis is controlled.[5] The fast and complete recovery of our patient may certainly be ascribed to the immediate and intensive treatment instituted, which resulted in a very rapid correction of acidosis.

The beneficial effect of hemodialysis in methanol poisoning is well documented.[6] The role of hemodialysis in the favorable outcome in our patient is hard to assess, but there is reason to believe that it most efficiently helped to remove toxic substances after the patient's kidneys failed to do so.

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Angiographic Demonstration of Fistula Between Abdominal Aorta and Thoracic Duct

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A traumatic fistula between the upper abdominal aorta and thoracic duct was observed and documented by means of abdominal aortography and selective celiac arteriography. Repeat aortography performed three weeks after the first examination demonstrated spontaneous closure of the fistula, with no persisting abnormalities.

PHYSICIANS responsible for the management of thoracic and abdominal trauma are becoming increasingly aware of the importance of angiographic assistance in the more precise evaluation of the nature and extent of injury. There have been, therefore, an increasing number of reports in modern medical literature describing the angiographic manifestations of trauma to the great vessels of the thorax and abdomen, the kidney, spleen, and liver. Although instances of thoracic duct injury have been reported in the surgical literature in the past, to our knowledge the precise angiographic diagnosis of such an occurrence resulting in fistulization between the thoracic duct and the abdominal aorta has never previously been documented.

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Report of a Case

A 35-year-old man was admitted to Mercy Hospital with serious injuries resulting from a motorcycle gang fight on Jan 19, 1969. There were multiple scalp lacerations and a stab wound in the left posterior side of the chest wall at the level of the tenth and 11th ribs. There was also a penetrating gunshot wound of the hemithorax on the lower right. The patient was hypotensive but not in shock. The hemoglobin level was 9 gm/100 cc and the hematocrit value was 26%. Roentgenograms of the chest revealed a bilateral hemothorax with a minimal left-sided pneumothorax.

The plain roentgenogram of the abdomen revealed moderate gastric dilatation but no indication of free air in the peritoneal cavity. The renal and psoas contours appeared undisturbed. The bilateral hemothorax was effectively managed by thoracocentesis and bilateral suction drainage. The patient's condition remained stable with the aid of intravenous fluids, several blood transfusions, and antibiotic coverage. In spite of the repeated blood transfusions, however, the hematocrit and hemoglobin levels remained low. Splenic injury resulting from the stab wound was clinically suspected and the patient was transferred to the Division of Radiology for celiac arteriography. With the Seldinger tech-

1. Abdominal aortogram demonstrating irregular fistulous tract (*white arrow*) between upper abdominal aorta and thoracic duct (*black arrow*) in area of cisterna chyli.